CLINICAL CASE REPORT

Delayed hyperbaric oxygen therapy for severe arterial gas embolism following scuba diving: a case report

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ABSTRACT

We present the case of a 42-year-old female who was critically ill due to an arterial gas embolism (AGE) she experienced while diving in Maui, Hawaii. She presented with shortness of breath and dizziness shortly after surfacing from a scuba dive and then rapidly lost consciousness. The diver then had a complicated hospital course: persistent hypoxemia (likely secondary to aspiration) requiring intubation; markedly elevated creatine kinase; atrial fibrillation requiring cardioversion; and slow neurologic improvement. She had encountered significant delay in treatment due to lack of availability of local hyperbaric oxygen (HBO_2) therapy.

Our case illustrates many of the complications that can occur when a patient suffers a severe AGE. These cases may occur even without a history of rapid ascent or risk factors for pulmonary barotrauma, and it is imperative that they be recognized and treated as quickly as possible with HBO_2. Unfortunately, our case also highlights the challenges in treating critically ill divers, particularly with the growing shortage of 24/7 hyperbaric chambers able to treat these ICU-level patients.

KEYWORDS: air embolism; arterial gas embolism; cerebral arterial gas embolism (CAGE); diving medicine; hyperbaric oxygen therapy

INTRODUCTION

Arterial gas embolism (AGE) is a relatively rare but potentially grave injury that may occur while scuba diving. These patients present fairly immediately after diving, with a spectrum of neurologic symptoms that can range from subtle mental status changes to unconsciousness with cardiovascular collapse. Diagnosis may be difficult, as the presentation can mimic other neurologic syndromes. The treating physician must maintain a high index of suspicion, as the only treatment is immediate hyperbaric oxygen (HBO_2) therapy. We present a case of severe AGE complicated by delay of diagnosis and lack of access to hyperbaric oxygen therapy.

HISTORY

The patient is a 42-year-old female recreational diver who was transferred from Maui, Hawaii, to our institution for treatment of presumed AGE following scuba diving.

Dives

On the day of injury the patient completed three dives, all on air, using a dive computer and making a safety stop at 15 feet on each dive. The first two were performed off the coast of Lanai: 63 feet for 43 minutes followed by a surface interval of 50 minutes, then a dive to 61 feet for 47 minutes followed by a two-hour surface interval. The last dive was off a wreck to 93 feet for 32 minutes. The diver's computer alarmed prior to ascent on the last dive. It was not recovered, and the reason is unknown. Her dive buddy denied observing any instances of emergency ascent, loss of buoyancy control, seizure activity, or coughing activity during the dives. No significant

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swimming effort was required during the dive, as there was minimal current, and no cold exposure.

The remainder of the divers from the same excursion were asymptomatic post-diving, including the dive buddy. The patient had a 30-year diving history without previous episodes of decompression illness (DCI). She had completed two dives without incident the day prior to injury.

Post-dive on the boat

Per the report from her dive buddy, the diver exited the water without issue, climbing the ladder onto the dive boat after the final dive. She removed her dive gear and was apparently asymptomatic until approximately 10-15 minutes after surfacing. At that time, as she was walking toward the aft of the boat, she reported becoming short of breath and "dizzy." There was no abnormal speech or weakness noted during this time. She indicated worsening shortness of breath and dizziness, fell to the deck, and then became unresponsive. Other divers reported a slightly different scenario, indicating that the diver seemed ataxic when exiting the water and felt dizzy within five minutes of surfacing and before she took off her equipment.

Transit time from the dive site to the dock where EMS was waiting was approximately 25 minutes. During this time, the diver received emergency surface oxygen with no CPR or airway support required. Right-sided gaze deviation and right-sided seizure activity were noted. Upon EMS assessment, atrial fibrillation with rapid ventricular response (RVR) was noted. She was intubated; her initial oxygen saturation post-intubation was reported to be 80%.

Arrival to the ER, testing

On arrival at the emergency room the patient was persistently hypoxic, with a partial pressure of arterial oxygen (P_aO_2) of 69 on mechanical ventilation and a fraction of inspired oxygen (F_iO_2) of 100%, and a positive end-expiratory pressure (PEEP) of 8. The patient remained unresponsive. She was given diltiazem for atrial fibrillation with RVR and showed no improvement. Cardiology completed a bedside echocardiogram that demonstrated mild left ventricular hypertrophy (LVH), as well as a reduced left ventricular ejection fraction (LVEF) of 35%-40%, hypokinesis of mid-apical cardiac segments, an appearance most consistent with stress-induced cardiomyopathy (Takotsubo). There was no evidence of LV thrombus. The patient was then given a bolus of 150 mg amiodarone and cardioverted with a single shock at 200 joules, with conversion to normal sinus rhythm at a rate of 100 beats per minute.

Initial neurologic exam of the patient while she was off sedation revealed reactive 4-mm pupils. Her eyes were held in mid-gaze when manually opened, and she did not open her eyes to stimulus. She had positive oculocephalic and cough reflexes. There was occasional purposeful movement of the left hand directed toward the face. Local right-sided seizure activity was noted overnight involving right deviated gaze, upper extremity and focal mild clonus to the diaphragm which resolved with the addition of propofol. She experienced hypotension that was not responsive to fluids and required the addition of a phenylephrine infusion to maintain blood pressure overnight. She was placed on midazolam and fentanyl for sedation, as well as antiepileptics levetiracetam and lacosamide.

Head computed tomography (CT)/computed tomography angiography (CTA) on the day of the injury revealed no acute findings. Chest X-ray revealed a normal cardiac silhouette and slight vascular congestion with interstitial edema.

The emergency room physician contacted a local undersea medicine practitioner who was initially told, in error, that a bedside echo revealed an intracardiac thrombus. Due to the new onset atrial fibrillation and this echo report, the presentation was initially felt to be cardioembolic in nature and not related to the patient's dives. The patient was admitted to the intensive care unit (ICU) and started on anticoagulation with heparin.

The next morning, the case was reviewed by the oncoming ICU physician, and the absence of an intracardiac thrombus was confirmed by cardiology. Brain magnetic resonance imaging (MRI) revealed no acute intracranial abnormality. CTA chest with contrast revealed no pulmonary embolism and no evidence of pneumothorax or pneumomediastinum. Electroencephalogram (EEG) revealed no active seizures but markedly attenuated activity, with a diffuse toxic/metabolic presentation while on midazolam, propofol, fentanyl, levetiracetam and lacosamide.

As the presentation was consistent with AGE, Divers Alert Network (DAN) was contacted. Hawaii lacked a hyperbaric chamber able to care for critically ill patients, so medical evacuation (MEDEVAC) arrangements to our institution were arranged. MEDEVAC required approximately 38 hours, including arrangements for appropriate aircraft, crew, and crew rest time.

While awaiting MEDEVAC, the patient was kept on an F_iO2 of 100%. She was noted to have increasing leukocytosis and fever. She was empirically started on piperacillin/tazobactam for presumed aspiration pneumonia. Troponins were elevated to a peak of 3.61 (ref <0.04 ng/mL), which was felt to be secondary to demand ischemia or Takotsubo cardiomyopathy. Repeat neurologic exam revealed no spontaneous movement, equal and reactive pupils, and normal cough and gag reflexes. Some repetitious myoclonic diaphragm movements as well as rhythmic movement of the right arm and shoulder were noted and resolved with increased sedation. The patient received two episodes of hemodialysis for oliguria, a mildly elevated creatinine, hyperkalemia of 6.8 (ref 3.3-5.1 mmol/L), and an elevated creatine kinase (CK) which peaked at 6632 (ref 48-240 U/L). Levels normalized without recurrence, urine output normalized and vasopressor support was gradually weaned prior to transport.

MEDEVAC to U.S. mainland and treatment

The patient was transferred via aircraft pressurized to 1 atmosphere absolute for HBO_2 therapy. She arrived at our institution sedated on propofol, midazolam and fentanyl. Upon arrival to the ICU, tympanostomy tubes were placed by ENT in anticipation of HBO_2 therapy. She remained unresponsive, with no spontaneous movement or movement in response to pain. She did not open her eyes to noxious stimuli or follow commands.

Approximately four hours after her arrival in the ICU, the patient was moved to the hyperbaric chamber. She had received 100% normobaric oxygen while awaiting MEDEVAC and 75% normobaric oxygen during MEDEVAC. Due to concerns of pulmonary oxygen toxicity, and the fact that it was approximately 61 hours post injury, she was treated on a U.S. Navy Treatment Table 6 (TT6) without extensions, for a duration of 298 minutes. The patient remained sedated, with no change in neurologic exam after the initial treatment. She was treated once daily with a U.S. Navy Treatment Table 9 (TT9) for the following three days.

A follow-up head CT three days post injury was compatible with multiple infarcts that were "likely embolic given the history." Further repeat CTs of the head demonstrated expected evolution of these lesions. MRI/MRA testing was completed to further characterize cerebral injury. These tests demonstrated focal acute infarct within the medial left occipital lobe associated with scattered, left greater than right, regions of gyriform diffusion restriction "likely related to acute ischemia on the basis of suspected barotrauma related to scuba diving."

Continuous EEG demonstrated moderate diffuse nonspecific abnormalities suggesting diffuse cerebral dysfunction; however, no electrographic seizures or epileptiform discharges were noted.

Repeat echocardiogram revealed normal ventricular size and function, with an ejection fraction of 65%, moderate pulmonary hypertension, and mildly dilated left atrium. Saline contrast bubble study was early positive, suggesting an intra-atrial shunt. A subsequent transesophageal echo (TEE) was completed and a small patent foramen ovale (PFO) was detected by color flow Doppler.

Due to increased secretions and oxygen requirements, the patient underwent bronchoscopy three days post incident. Some thick secretions were suctioned to clear the right upper, right lower and left lower lobes, which improved her oxygenation and ability to wean F_iO_2.

Patient improvement

Sedation was progressively decreased by the ICU team after the third HBO_2 treatment. The patient was successfully extubated the day after the fourth HBO_2 treatment (seven days post injury). We attempted to treat the patient after extubation, but she requested discontinuation of treatment after the first oxygen period due to anxiety and frustration with duration of treatment. She refused additional hyperbaric treatments thereafter. Due to the aspiration pneumonia and fluid overload, she was diuresed, completed a course of vancomycin and ceftazidime, and was successfully weaned to room air.

The patient's neurologic exam progressively improved. Nine days after the incident, the patient was discharged from our hospital to a rehabilitation center. At that time, she was oriented to person and place, but she believed the year to be 2013. She had mild left upper-extremity and bilateral lower-extremity weakness and was able to walk 14 steps with a front-wheeled walker. Follow-up assessment with neurology at 78 days post injury showed complete resolution of symptoms and a normal neurologic exam.

DISCUSSION

This severe case of AGE highlights important clinical features of a relatively rare neurologic syndrome, as well as the challenges of treating patients who are affected.

AGE can occur through a variety of mechanisms. In diving, it is typically secondary to pulmonary barotrauma (PBT) with the subsequent entrance of gaseous emboli into the pulmonary vasculature. This is often associated with a history of rapid ascent, breath-holding, cough or Valsalva maneuvers, though there are cases of AGE occurring in a seemingly "uneventful" ascent, as with our patient. These bubbles may travel to the left atrium, then to the left ventricle and subsequently to the aorta and out to systemic circulation. Cardiac shunts, such as a PFO, can facilitate the passage of venous bubbles directly into the systemic arterial circulation.

The two main organs affected by AGE are the brain and heart. Arterial bubbles cause distal ischemia with resultant breakdown of the blood-brain barrier and subsequent cerebral edema. Bubbles occlude small arterioles with diameters of 30-60 μm which are found at the junction between white and gray matter in the brain [1]. Neurologic deficit distribution can be highly varied due to the random spread of bubbles in the cerebral vasculature. Symptoms of AGE include unconsciousness, paralysis, paresthesias, vertigo, convulsions, vision changes, confusion and seizures. The common axiom in diving is that any diver with loss of consciousness within 10 minutes of surfacing from a dive has an AGE until proven otherwise [2].

Although AGE is a clinical diagnosis, there has been a correlation between elevated creatine kinase and AGE. Our patient's significantly elevated CK is consistent with previously published values seen in patients with severe AGE. In this same article, it was observed that higher values of CK were correlated with worse neurologic outcomes, though not necessarily with their initial neurologic presentation [3]. Chest X-ray findings may show pneumothorax as well as pulmonary edema from cardiac dysfunction [4]. Even without evidence of pulmonary barotrauma, these patients may present with severe hypoxemia due to aspiration [3]. In one case series of AGE, radiographic findings of pulmonary barotrauma was present in only 42% of patients [4].

Our case illustrates the important point that unlike other stroke syndromes (e.g., ischemic, hemorrhagic, cardioembolic), AGE may not have findings that are immediately evident on radiographic imaging (including CT and MRI). When findings do become evident on MRI, they may show extensive gray matter changes and restricted diffusion. As was the case in our patient, there are often discrepancies between timing of symptoms and radiographic findings [5]. It must be emphasized that initial brain imaging is often normal, even in the setting of severe neurological deficits, though occasionally intravascular air may be detected. As such, diagnosis of AGE should not depend on imaging results, nor should it delay HBO_2 therapy in cases highly suggestive of AGE [6]. Another significant difference observed is that while these patients may present with severe neurologic deficits, they will often show dramatic clinical improvement after three to five days of treatment, generally not seen in other stroke syndromes [7,8].

There are elements of our case that are not completely typical of AGE. First, the source of the arterial gas emboli in our case is unclear. The inciting dives were non-decompression dives, though they did approach no-decompression limits. The patient's dive computer alarmed prior to the commencement of ascent. It is unknown, however, whether this was due to decompression obligation or ascent rate. The patient cannot recall the events surrounding her last dives. There was no known emergency ascent, loss of buoyancy, or coughing underwater as noted by the dive buddy, and no noted PBT on CT chest. However, PBT can occur in as little as 4 feet of water and may go unnoticed by a dive buddy. PBT may not be detectable on CT [9].

Second, as mentioned above, the typical time course for symptoms of an AGE is almost immediately upon surfacing, or at least within 10 minutes. Per the patient's wife, who was on the dive boat, there was a delay of about 15 minutes prior to the onset of symptoms. There are potentially two explanations for this occurrence. The first is that the wife is wrong about the time course of the symptoms. Other divers on the boat reported that the patient seemed ataxic as soon as she exited the water, which would be more consistent with an AGE. It is possible that the patient's wife did not notice this and the patient did not report symptoms immediately.

Another possible explanation for the delay is a paradoxical gas embolism, or a venous embolism that travels to the arterial circulation. Our patient experienced shortness of breath prior to loss of consciousness, new onset atrial fibrillation and was subsequently diagnosed with a PFO. Divers may form venous gas bubbles/emboli (VGE) even when following "safe" dive practices [10].

A series of gas bubbles may enter the microcirculation of the lung, causing tissue damage, shortness of breath, pulmonary edema and acute respiratory distress syndrome. VGE that pass through the lungs may enter the arterial circulation as AGE, be dispersed to coronary arteries and the cerebrum, and result in possible tachydysrhythmias, ventilation-perfusion mismatch, and neurologic deficits [11]. Elevated right atrial pressure secondary to VGE-induced elevation of pulmonary arterial pressure can allow right-to-left flow through a PFO [12]. This elevated pressure may also induce atrial fibrillation.

In addition, boarding the boat wearing full gear after diving or straining while removing or moving equipment could be sufficient to alter intrathoracic pressure, thus promoting right-to-left shunting of blood through a PFO. This may potentially explain why there was supposedly a delay of approximately 15 minutes to onset of symptoms [13].

Benson, et al. retrospectively reviewed iatrogenic AGE cases at Hennepin County between 1987-1999. All nine cases that had a venous source of AGE suffered from pulmonary symptoms in addition to neurologic signs or symptoms. Conversely, none of the 10 individuals with an arterial source of AGE experienced pulmonary symptoms. Eight of the venous cases revealed pulmonary edema as shown on chest X-rays, and six demonstrated cardiac arrhythmias or ischemic changes [11].

A gas embolism in the coronary arteries can cause cardiac dysfunction, ECG changes and arrhythmias from ischemia [14]. It has also been observed that isolated AGE can cause ECG changes, even in the absence of coronary artery gas [15]. Based on the totality of her presentation, we hypothesize that the cause of the atrial fibrillation on our patient was secondary to AGE and possible gas in her coronary arteries and unlikely undiagnosed atrial fibrillation. She also suffered from a stress-induced cardiomyopathy (Takotsubo), which has been seen in diving-related illnesses [16].

We considered other cardiopulmonary causes of her symptoms as well, including immersion pulmonary edema (IPE) or a primary cardiac event. However, the lack of symptoms during the dive, as well as associated neurologic symptoms make IPE an unlikely cause of her illness. It is possible that the patient could have suffered an acute cardiovascular event such as a myocardial infarction or arrhythmia. However, this would not explain her neurologic deficits and would be unlikely to cause such significant hypoxia and multisystem organ damage.

Hyperbaric chamber availability

Although this patient survived a potentially catastrophic injury with a good outcome, this case highlights some of the challenges presented in treating this type of injury. Our patient was diving in a location that until recently had the capabilities of treating critically injured divers. The loss of hyperbaric chambers available to treat divers on an emergent, 24/7 basis is not unique to Hawaii. In the 1990s, there were more than 200 facilities in the DAN referral network. As of 2016 there are only 91, 43 of which are capable of 24/7 emergency care.

On the mainland of the United States the average distance to transport a patient is 96 miles [17,18]. We suspect that most recreational divers in Hawaii are not aware that there is no longer a hyperbaric chamber available to treat them if they are significantly injured. This has the potential to affect the islands' tourist industry, a significant source of revenue, as well as the safety of its employees in the dive industry. As highlighted by this case, this loss in Hawaii significantly delays patients' time to treatment.

Currently the closest chamber available to treat critically injured divers in Hawaii is our program in California, a distance of approximately 2,500 miles. The impact and significant financial cost of this evacuation should not be underestimated. A fixed-wing aircraft from the mainland, with an experienced flight crew and physician, is required to evacuate a critically ill patient for this distance. The process to arrange this takes significant time and may be affected by plane and crew availability, as well as by weather conditions. There is risk to both the patient and the flight crew in the transport process. Fortunately for our patient the evacuation was arranged by DAN, but despite their best efforts, treatment was still significantly delayed.

Aside from the potential transport risks, perhaps the most significant negative impact on a patient is the delay to treatment with HBO_2. It is well established that HBO_2 treatment for AGE and decompression sickness should be initiated as soon as possible, as shorter time to treatment has been shown to have a favorable impact on outcome [19,20]. This is not to say that the HBO_2 should not be provided if a delay to treatment is unavoidable: Improvements have been reported in treatments with delays of 39-60 hours, but these delays should be avoided if at all possible [7].

CONCLUSIONS

Arterial gas embolism is a potentially serious complication of diving and a major cause of morbidity and mortality. It is imperative that AGE be diagnosed quickly and treated immediately with hyperbaric oxygen therapy. Complications include permanent neurologic deficits, cardiac arrhythmias, cardiomyopathies, aspiration pneumonia and acute kidney injury, all of which were experienced by our patient. Unfortunately, delays to treatment and worsening outcomes of patients may occur as the number of hyperbaric chambers capable of treating critically ill patients continues to decline.

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